Eukaryotic cells can be compared to mobile phones. Both are small and powerful and can work as a stand-alone system or easily interact with others. Each mobile phone consists of subassemblies that have well-defined functions and efficiently co-operate with each other. Those subassemblies in eukaryotic cell are called organelles and are essential for its proper functioning. Among these, mitochondria are the most unusual ones. They act like a self-charging battery that provides energy for the cell and to maximize their productivity, mitochondria have specific structure. They consist of two membranes: outer and inner. The outer membrane fully surrounds the inner membrane like a rubber bumper that completely covers the mobile phone from all sides. Contrary to smooth outside of the mobile phone that is fitted around by the bumper, the mitochondrial inner membrane folds many times and creates layered structures. As a result the two membranes are not in direct, physical contact and a small intermembrane space is formed in between. The inner membrane surrounds the mitochondrial matrix and those two components act together to produce the energy for the cell.

Another feature that makes mitochondria special is the presence of their own DNA. Although the size of mitochondrial DNA is not as large as the nuclear one, it encodes 13 key components in energy production process. However, for mitochondria to be fully active, it requires more than a 1000 proteins. The remaining 99% of mitochondrial proteins are encoded by the nuclear DNA and need to be imported into mitochondria. This process is not easy due to the complex mitochondrial structure and requires special machinery that helps proteins to get to their functional destinations in mitochondria. Disorders in protein import can cause mitochondrial dysfunctions that have serious consequences for the cell and are associated with different diseases. Majority of mitochondria related diseases affect the brain and muscles, due to their high energy requirements. Alzheimer's disease is currently the most common form of dementia, affecting approximately 46 million people worldwide and it is estimated that by 2050, the number of cases will triple.

The main task is to understand how the proteins are imported into the mitochondria. Currently, we know a lot about the system that helps to transport mitochondrial proteins, but there are still some gaps that prevent us from seeing the full picture. For example, it is not known what is really happening at the cytosolic stage of the mitochondrial transport and whether all proteins are transported after completing the synthesis in the cytosol or for some of them the synthesis is coupled with the import into the mitochondria. To address these questions, I am going to investigate the protein import into mitochondria using zebrafish as a model organism. Zebrafish is a small tropical fish that shares many similarities with human, including having the same major organs and tissues. Moreover, human and zebrafish share 70% of genes and 84% of genes known to be associated to human pathologies has a counterpart in zebrafish. Those features ensure that information acquired through zebrafish is more accurate than obtained by *in vitro* studies, thus easier to extrapolate to human biology.

Since the import of mitochondrial proteins is essential to functioning of mitochondria, we hope that accurate understanding of this process will contribute to prevention strategies against mitochondrial dysfunctions induced diseases.